Using the Amygdala in decision making

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April 15, 2015

Decision making is often described as composed of multiple loops, mainly the limbic, associative, and motor loops, in the Prefrontal Cortex and Basal Ganglia. While the various nuclei of the Amygdala has been traditionaly considered for their role in fear prediction and respondent conditioning [9, 4, 7], structural similitudes have been reported between the central amygdala (CeA) and structures involved in decision making the nucleus accumbens and the pallidum [5]. Particularly, the lateral capsular, lateral and medial subdivisions of CeA possess similarities in structures and connectivity respectively with the shell, the core of the nucleus accumbens and the pallidum. This, along with a spatial continuity between CeA and the shell of the nucleus accumbens [5], leads to the hypothesis that respondant conditioning could be seen as a loop more primitive but similar to decision-making loops. Moreover, lesions of the amygdala, and especially of the basal nucleus of the amygdala, impair operant conditioning paradigms like devaluation or reversal [8], or decision making in gambling [1]. In a direct way, learning associations between CS (conditioned stimuli) and US (unconditioned stimuli, ie. reward or punishment) allows the amygdala to learn CS values [2], and to provide such values in to the OFC and ventral striatum for goal-directed behaviors [8]. In an indirect way, the amygdala projects to VTA-SNc for dopamine and to the basal forebrain for acetylcholine, thus providing indirect reinforcing signals to the decision making system.

We present here a simple neuronal model of the amygdala and propose to compare it to the decision making loops. Our model is composed of five populations from three different amygdalar nuclei. Neurons in these populations are described using a classical mean-rate formalism and a sigmoid activation function. Learning is hebbian and uses a Rescorla-Wagner like prediction error. One specificity is that amygdalar activation also takes into account the effect of acetylcholine, which modulates the competition between different amygdalar populations for chosing a sensory-based rule or a contextual one [11, 10]. Acetylcholine concentration is computed from the recent prediction errors of our model, and as such reflects the known uncertainty in reward prediction [12]. This model successfully reproduces experimental data recorded in fear and extinction conditioning [7], along with the effect of pathways impairment as reported in [11, 10, 3].

This model is the first step in modeling operant conditioning and goal-directed behavior. Thus, ongoing work is to extend this model to operant conditioning, by including OFC, shell and ventral pallidum structures. Another work in progress proposes to use the uncertainty level computed by our amygdalar network to help a decision making system to choose between exploration and exploitation. If known uncertainty is low, it means the model is correctly predicting the rule, so it should favour exploitation. At the opposite, the higher the uncertainty in predicting US, the more should it explore, because its current strategy is not reliable.

We propose to highlight here both the similarities in functioning and reciprocal influences between respondent conditioning, as performed by our amygdalar model, and decision making.

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